after BMT. Thus, the data suggest that alteration in UC function plays a role in modifying liver-lung interaction during sepsis and acute lung injury.

Please replace the paragraph beginning at page 115, line 10, with the following rewritten paragraph:

The T1405N allele exhibits 50% heterozygosity and appears to be a silent variant in normal healthy adults. However, consequences of the qualitative change can be unmasked by stressful conditions. As disclosed in Examples 1-3, studies on adults exposed to high-dose chemotherapy in preparation for bone marrow transplantation demonstrated that the threonine-containing enzyme produces inadequate levels of arginine and citrulline and is associated with an increased incidence of hepatic veno-occlusive disease, acute lung injury, and death. As nitric oxide (NO) is generated in endothelial cells from L-arginine by nitric oxide synthetase (NOS), decreased levels of urea cycle intermediates could predispose to disturbances in vascular tone by limiting endogenous NO production.

Please delete the current pending sequence listing and insert in place thereof the sequence listing attached hereto.

IN THE CLAIMS:

Please cancel claims 10-14 and 21-25.

Please amend claim 1 as follows:

1. (Once Amended) A method of treating or preventing sub-optimal urea cycle function in a human subject having a polymorphism that results in a N→T substitution at amino acid 1405 of a carbamyl phosphate synthetase I (CPSI) polypeptide, the method comprising administering to the human subject a therapeutically effective amount of a nitric oxide precursor, whereby treatment or prevention of sub-optimal urea cycle function is accomplished.

Please amend claim 15 as follows:

15. (Once Amended) A method of treating or preventing bone marrow transplant toxicity in a human subject undergoing bone marrow transplant, the human subject's genome having at least one copy of a carbamyl phosphate synthetase I (CPSI) gene that encodes a threonine residue at amino acid 1405 of a CPSI polypeptide, the method comprising administering to the human subject a therapeutically effective amount of a nitric oxide precursor, whereby bone marrow transplant toxicity is treated or prevented in the human subject.

REMARKS

I. Status Summary

Claims 1-25 are pending in the present U.S. patent application.